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Prevalence and Severity of Circumflex Coronary Artery Disease in Electrocardiographic Posterior Myocardial Infarction

EDWARD W. BOUGH, MD, FACC, KENNETH S. KORR, MD, FACC

Providence, Rhode Island

To determine the coronary anatomy responsible for electrocardiographic posterior myocardial infarction, the prevalence and severity of disease in the right coronary and left circumflex coronary arteries were compared in 21 patients with electrocardiographic posterior infarction (17 of whom had associated inferior infarction) and 23 patients with isolated electrocardiographic inferior infarction. Significant circumflex coronary artery disease (≥75% stenosis) was more prevalent in patients with posterior or inferoposterior infarction (17 of 21) than in those with isolated inferior infarction (11 of 23) ($p < 0.02$). Significant right coronary artery disease was less prevalent in patients with posterior or inferoposterior infarction (16 of 21) than in those with isolated inferior infarction (23 of 23) ($p < 0.05$). Among the 21 patients with posterior or inferoposterior infarction, disease was more severe in the circumflex coronary artery in 10 and the right coronary artery in 5 and was of equal severity in 6. Among the 23 patients with isolated inferior infarction, the more diseased artery was the right coronary artery in 21 and the circumflex artery in 2 ($p < 0.001$ by chi-square analysis). Variant patterns of coronary artery dominance accounted for only 4 of the 17 patients with inferoposterior infarction. These data suggest that the likely substratum for electrocardiographic posterior or inferoposterior infarction is severe circumflex coronary artery disease, usually in association with significant right coronary artery disease. The pattern of tall, wide R waves in leads V_1 or V_2 ($RV_{1,2}$) in patients with inferior infarction is highly predictive of at least two vessel coronary artery disease.

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It has been recognized for almost 40 years that tall, wide R waves in leads V_1 or V_2 ($RV_{1,2}$ pattern), in the absence of right ventricular hypertrophy, may be diagnostic of myocardial infarction (1). Although such infarctions are called posterior or true posterior in current electrocardiographic terminology (2), their exact location and the associated distribution of coronary artery disease have not been well established.

Several pathologic studies (3,4) have shown that electrocardiographic posterior infarction is not associated with necrosis of the basal inferior left ventricular wall, which has been a long-standing clinical assumption (5). More recently, there has been reported (6) compelling mathematic and radionuclide evidence that electrocardiographic posterior infarction is primarily associated with asynergy and presumed necrosis of the basal lateral left ventricular wall, an area supplied primarily by the circumflex coronary artery. In addition, recent studies of patients with isolated left circumflex coronary artery disease (7) and patients with isolated lateral wall infarction (8) suggest a strong association between lateral wall infarction, significant circumflex artery disease and the electrocardiographic $RV_{1,2}$ pattern of so-called posterior infarction.

The purpose of this study, therefore, was to test the hypothesis that the prevalence and severity of circumflex coronary artery disease are greater in patients with electrocardiographic posterior or inferoposterior infarction than they are in control patients with only electrocardiographic inferior infarction.

Methods

Patients. The subjects of our study were 44 retrospectively selected patients who had undergone diagnostic coronary arteriography and who had contemporaneous electrocardiographic evidence of isolated posterior infarction (4 patients), inferoposterior infarction (17 patients) or isolated inferior infarction (23 patients). Patients were not included if they had left ventriculographic evidence of anterior infarction; Q waves in leads V_1–V_6, I or aVL; right bundle branch block; or clinical or catheterization evidence of right ventricular hypertrophy.

From the Division of Cardiology, The Miriam Hospital and Brown University, Providence, Rhode Island.

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Address for reprints: Edward W. Bough, MD, The Miriam Hospital, 164 Summit Avenue, Providence, Rhode Island 02906.

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Electrocardiographic criteria. Tall, wide right precordial R waves (subsequently referred to as $RV_{1,2}$ pattern) are the scalar electrocardiographic hallmark of posterior myocardial infarction. For R waves of 0.04 second or more and greater than or equal to S, it has recently been shown that RV_{1+2} is the most specific but least sensitive criterion for posterior infarction, that RV_2 is the most sensitive but least specific criterion and that RV_2 + a Q wave in inferior leads (leads III and aVF) is intermediate in specificity and sensitivity (6). RV_1 without an R wave in lead V_2 is extremely uncommon (6).

The 21 patients with posterior or inferoposterior infarction were distributed according to electrocardiographic criteria as follows: 1) RV_{1+2} (12 patients); 2) RV_2 (2 patients); and 3) RV_2 + Q wave in inferior leads (7 patients).

Criteria for inferior infarction were Q waves greater than 0.03 second as suggested by Horan et al. (9) with the usual Minnesota code modification (10) to increase the specificity for lead III: 1) lead II: Q greater than 0.03 second; 2) lead III: Q greater than 0.03 second and Q greater than or equal to 1 mm in lead aVF; and 3) lead aVF: Q greater than 0.03 second. Unless specifically stated otherwise, the term posterior infarction will be used to describe patients with either inferoposterior infarction or isolated posterior infarction and the term inferior infarction will be used to describe patients with isolated inferior infarction.

Coronary arteriography and grading of disease severity. All patients underwent percutaneous arterial catheterization and coronary arteriography in a fasting, mildly sedated state. Multiple views of both coronary arteries, including hemiaxial views of the proximal left coronary system, were always obtained. Coronary artery stenoses were graded and labeled according to standard American Heart Association recommendations (11). Stenoses were graded as 25, 50, 75, 90, 95, 99 or 100% reduction in luminal diameter and were localized to the proximal, middle or distal segments of the three coronary arteries and their major branches: diagonal, marginal or posterior descending arteries. A vessel was considered to have significant disease if there was 75% or more stenosis of the main artery or a major branch.

Comparative severity of atherosclerotic disease in the left circumflex and right coronary arteries was determined from three criteria: 1) severity of luminal diameter reduction by single or multiple stenoses; 2) extent of vascular distribution jeopardized by stenoses; and 3) direction of intercoronary collateral flow, if present. For example, a left circumflex artery with a 90% proximal stenosis was considered more severely diseased than a right coronary artery with a 50% proximal stenosis, but a right coronary artery with a 75% proximal stenosis was considered more severely diseased than a circumflex artery with only a 90% stenosis in a small marginal branch. When severity of disease in the two arteries appeared equal on the basis of stenoses and vascular distribution compromised, the direction of intercoronary collateral flow, if present, was used to identify the more severely diseased artery.

Radionuclide angiography. Gated radionuclide ventriculograms, obtained in 38 of the 44 patients, were acquired in a 64 × 64 matrix at 24 frames/cycle and 250,000 counts/frame in both the anterior and caudally angulated (30°) left anterior oblique projections. Ejection fraction images were generated for both projections by a modification of the method of Maddox et al. (6,12). The caudally angulated left anterior oblique projection is almost perpendicular to the long axis of the left ventricle and provides an excellent tangential view of the lateral wall from apex to base. Nine left ventricular segments (Fig. 1) were graded as normal = 1, hypokinetic = 2 and akinetic or dyskinetic = 3, based on blinded review of loop movies and ejection fraction images.

Statistical analysis. Differences between small proportions were assessed for significance by Fisher's exact test as compiled for 2 × 2 contingency tables (13). Row by column contingency tables were studied by chi-square analysis with a commercially available statistical package (Human System Dynamics) on an Apple II Plus microcomputer.

Results

Prevalence of left circumflex and right coronary artery disease. Table 1 provides a detailed summary of the electrocardiographic and coronary arteriographic findings in patients with inferior infarction and with posterior or infero-

Figure 1. Schematic diagram of the nine left ventricular segments identified and scored for the anterior (ANT) and caudally angulated left anterior oblique (LAO) radionuclide projections. MV = mitral valve.

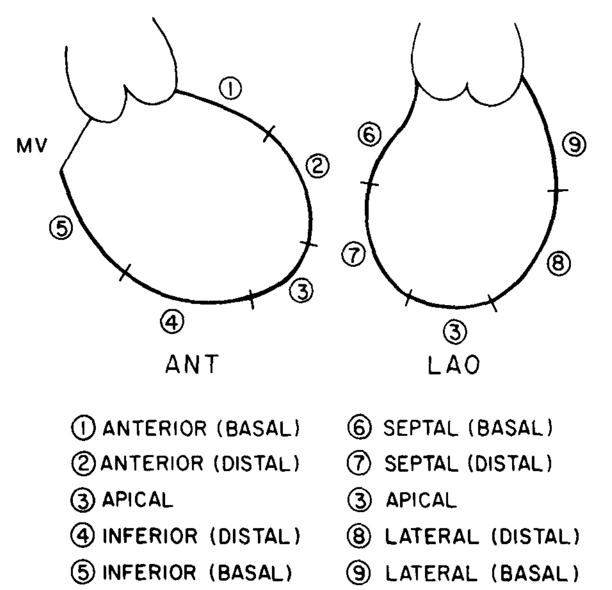

Table 1. Coronary Angiographic, Electrocardiographic and Radionuclide Data in 44 Patients

	Coronary Anatomy						ECG		RVG		
	Angiographic Degree of Stenosis								LV Segmental Motion Scores		
Case	LAD	LCx	RCA	Collateral Vessels	Coronary Artery Dominance	Artery (LCx versus RCA) With Greater Disease	Q Wave > 0 03 Second	$RV_{1,2}$ or RV_2	4	5	9
				Posterior/Inferoposterior Infarction							
1	Normal	99% Prox 99% OM-1	75% Prox, 50% dist	RCA → OM-1	Right	LCx	III, aVF	$V_1 + V_2$	3	3	3
2	90% Prox, 75% mid, 90% D-1	100% Mid, 50% OM-1	75% Prox	LCA → OM-2, PDA	Codominance	LCx	II, III, aVF	$V_1 + V_2$	1	2	2
3	99% Mid	99% Prox, 99% OM-1	99% Prox	Prox RCA → LAD, LAD → PDA	Right	Equal	II, III, aVF	$V_1 + V_2$	1	1	3
4	Normal	100% Prox	90% Prox	0	Left	LCx	II, III, aVF	$V_1 + V_2$	2	2	3
5	Normal	Vestigial	50% Prox, 99% dist	0	Right (lateral LV branch)	RCA	II, III, aVF	V_2	2	2	3
6	Normal	99% OM-1	25% Prox	RCA → OM-1	Codominance	LCx	II, III, aVF	$V_1 + V_2$	3	2	2
7	Normal	99% Prox	Normal	0	Right	LCx	0	$V_1 + V_2$	2	2	3
8	50% Dist, 50% D-1	90% Prox, 100% mid	75% Mid, 90% dist	LAD → OM-2, 3	Right	LCx	II, III, aVF	$V_1 + V_2$	2	2	3
9	100% Mid	100% Prox	100% Prox	Prox LAD → PDA, OM-1, 2, Prox RCA → LAD, OM-2	Right	Equal	II, III, aVF	V_2	3	3	2
10	99% Mid, 99% D-1	95% OM-1	90% Prox	LCx → dist RCA, PDA	Right	Equal	II, III, aVF	V_2	—	—	—
11	Normal	Normal	99% Mid	0	Right	Right	0	V_2	1	1	1
12	75% D-1, 2	50% OM-1	100% Prox	LCx → dist RCA, PDA	Right	Right	III, aVF	V_2	2	3	1
13	Normal	100% Prox, 100% OM-1	100% Prox	LAD → dist RCA, PDA, prox LCx → OM-2, 3	Right	Equal	II, III, aVF	V_2	1	3	2
14	75% Mid	99% Mid	50% Mid, 50% dist	0	Right	LCx	0	$V_1 + V_2$	2	3	2
15	Normal	99% Mid, 99% OM-1	90% Mid	LAD → PDA	Right	LCx	0	V_2	3	3	1
16	75% Mid	99% Prox	50% Dist	0	Right	LCx	II, III, aVF	$V_1 + V_2$	1	1	1
17	Normal	100% OM-1, 2	99% Prox	LAD → OM-1, 2, PDA	Right	Equal	III, aVF	V_2	1	1	2
18	90% Mid	100% Prox	50% Mid, 50% prox	RCA → mid LCx, OM-1, 2	Right	LCx	II, III, aVF	$V_1 + V_2$	2	2	3
19	Normal	Normal	100% Prox	LAD → PDA	Right	RCA	II, III, aVF	$V_1 + V_2$	3	3	1
20	Normal	99% Mid	100% Prox	LAD → PDA, prox LCx	Right	RCA	II, III, aVF	$V_1 + V_2$	1	2	1
21	75% Prox, 75% mid	99% OM-1	100% Prox, 100% mid	LAD → OM-1, PDA, prox RCA → dist RCA, PDA	Right	RCA	II, III, aVF	V_2	2	3	3
				Inferior Infarction							
22	90% Mid	25% OM-1	90% Prox, 100% mid	Prox RCA → dist RCA	Right	RCA	III, aVF	0	1	2	1
23	99% Mid	99% OM-1	100% Mid	Prox RCA → PDA, LCx → dist RCA, PDA	Right	RCA	III, aVF	0	3	2	1
24	Normal	50% OM-1	99% PDA	LCA → PDA	Right	Right	II, III, aVF	0	2	3	1
25	50% Prox	Normal	99% Prox, 50% mid	LCA → PDA	Right	Right	III, aVF	0	2	2	1

Table 1 (*continued*)

	Coronary Anatomy						ECG		RVG		
	Angiographic Degree of Stenosis								LV Segmental Motion Scores		
Case	LAD	LCx	RCA	Collateral Vessels	Coronary Artery Dominance	Artery (LCx versus RCA) With Greater Disease	Q Wave > 0.03 Second	$RV_{1,2}$ or RV_2	4	5	9
				Inferior Infarction							
26	90% Mid	Normal	100% Prox	Prox RCA → dist RCA	Right	Right	II, III, aVF	0	3	3	1
27	100% Mid	99% Prox	100% Mid	Prox RCA → LAD	Right	Right	III, aVF	0	3	3	1
28	70% D-1	Normal	100% Prox	0	Right	Right	II, III, aVF	0	3	3	1
29	100% Prox	Normal	50% Prox, 75% dist, 90% PDA	Prox RCA → LAD, LCx → LAD	Right	Right	III, aVF	0	2	2	1
30	50% Mid, 75% D-1	100% Mid, 100% PDA	100% Prox, (nondominant)	RCA → LCx	Left	LCx	II, III, aVF	0	2	3	1
31	99% Prox, 99% mid	90% Mid	100% Prox, 100% mid	Prox RCA → dist RCA, LCA → dist RCA	Right	Right	III, aVF	0	—	—	—
32	Normal	Normal	100% Prox, 100% mid	LCA → dist RCA, PDA	Right	RCA	III, aVF	0	—	—	—
33	99% Mid	75% OM-1, 95% OM-2	50% Mid, 75% dist, 100% PDA	0	Right	RCA	III, aVF	0	3	3	1
34	Normal	100% OM-1	99% PDA	LAD → OM-1	Right	LCx	III, aVF	0	—	—	—
35	Normal	Normal	100% Prox, 100% mid	LCA → dist RCA, PDA	Right	RCA	II, III, aVF	0	3	3	1
36	99% Mid, 90% D-1	99% Prox, 50% mid	90% Prox, 100% mid	Prox RCA → dist RCA	Right	RCA	III, aVF	0	3	3	3
37	90% Prox, 90% D-1	90% Mid, 90% OM-2	100% Prox, 100% mid, 75% dist	LAD → PDA	Right	RCA	III, aVF	0	1	2	1
38	80% Prox, 95% mid	Normal	100% Prox, 100% mid	LCA → dist RCA, PDA	Right	RCA	II, III, aVF	0	—	—	—
39	99% Mid, 90% D-1, 75% D-2	50% OM-1	99% Dist	LCA → dist RCA	Right	RCA	III, aVF	0	—	—	—
40	50% Dist	75% Prox, 90% OM-1	100% Prox, 100% mid	LCx → dist RCA	Codominance	RCA	II, III, aVF	0	3	2	1
41	75% Prox	Normal	50% Prox, 90% dist	0	Right (lateral LV branch)	RCA	III, aVF	0	3	3	1
42	Normal	75% Mid, 75% OM-1, 2	50% Prox, 99% mid, 100% dist, 100% PDA	LCA → dist RCA, PDA	Right	RCA	III, aVF	0	2	2	1
43	75% Prox, 90% mid	50% Prox, 90% mid	100% Prox, 100% mid	LCA → PDA	Right	RCA	III, aVF	0	2	2	3
44	Normal	Normal	100% Prox, 100% dist	LCA → dist RCA	Right	RCA	III, aVF	0	3	3	1

D = diagonal artery; dist = distal; ECG = electrocardiogram, LAD = left anterior descending coronary artery; LCA = left coronary artery; mid = middle; LCx = left circumflex coronary artery, LV = left ventricular, OM = obtuse marginal artery; PDA = posterior descending artery, prox = proximal, RCA = right coronary artery, RVG = radionuclide ventriculography; left ventricular segments from Figure 2. 4 = distal inferior, 5 = basal inferior, 9 = basal lateral posterior infarction. Comparisons of the prevalence of significant (≥75% luminal stenosis) disease in the left circumflex and right coronary arteries for patients with electrocardiographic posterior and inferior infarction are presented in Figure 2.

In the 21 patients with electrocardiographic posterior or inferoposterior infarction, the prevalence of disease in the circumflex (n = 17) and the right (n = 16) coronary artery was nearly equal (Fig. 2, comparison A), whereas in the 23 patients with electrocardiographic inferior infarction, right coronary artery disease was significantly ($p < 0.01$) more prevalent (n = 23) than circumflex artery disease (n = 11) (Fig. 2, comparison C).

From the viewpoint of arterial involvement, circumflex artery disease was significantly ($p < 0.02$) more prevalent in patients with posterior or inferoposterior infarction (17 of 21) than in those with inferior infarction (11 of 23) (Fig. 2, comparison B). Right coronary artery disease, by contrast, was significantly ($p < 0.05$) less prevalent in patients with posterior or inferoposterior infarction (16 of 21) than in those with isolated inferior infarction (23 of 23) (Fig. 2, comparison D).

Comparative severity of circumflex and right coronary artery disease. On the basis of previously presented methodologic criteria, an observer blinded to the electrocardiographic data analyzed the coronary arteriograms for each patient as showing more severe disease in the left circumflex or right coronary artery or equally severe disease in both arteries. In the 21 patients with electrocardiographic posterior or inferoposterior infarction, the more diseased artery was the circumflex artery in 10 patients and the right coronary artery in 6; the arteries were equally diseased in 5 patients (Table 2). By contrast, in patients with isolated electrocardiographic inferior infarction, the right coronary artery was almost exclusively (21 of 23) the more diseased artery. This difference in distribution of comparative severity of arterial disease for patients with isolated inferior

Figure 2. Comparisons of the prevalence of significant (>50% stenosis) disease in the left circumflex coronary artery (LCx) and right coronary artery (RCA) in patients with posterior or inferoposterior infarction (PMI) and those with isolated inferior infarction (IMI). Levels of significance from Fisher's exact test.

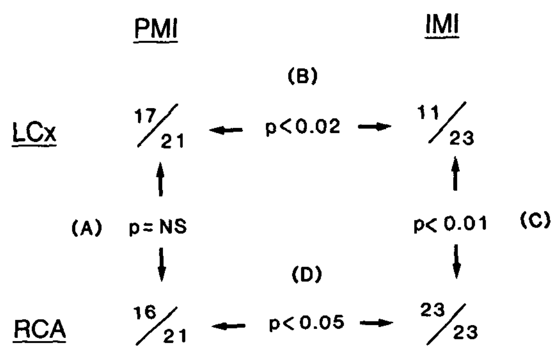

Table 2. Comparison of the Severity of Disease in the Left Circumflex and Right Coronary Arteries in Patients With Posterior or Inferoposterior and Isolated Inferior Myocardial Infarction

	Artery With More Severe Disease		
	LCx	Disease Equal in Both Arteries	RCA
Posterior/inferoposterior MI	10	5	6
Inferior MI ($p < 0.001$)*	2	0	21

*From chi-square analysis of distribution in 2 × 3 nonparametric contingency table. LCx = left circumflex coronary artery; MI = myocardial infarction; RCA = right coronary artery.

or posterior/inferoposterior infarction was highly significant ($p < 0.001$) by chi-square analysis.

Left ventricular asynergy. Radionuclide wall motion data are presented in the last column of Table 1. Among the 38 patients who underwent radionuclide ventriculography, inferior wall asynergy (segments 4 and 5) was equally prevalent in patients with posterior or inferoposterior infarction (16 of 20) and isolated inferior infarction (18 of 18) (p = NS). By contrast, basal lateral wall asynergy (segment 9) was far more prevalent in patients with posterior or inferoposterior infarction (14 of 20) than in those with isolated inferior infarction (2 of 18) ($p < 0.005$).

Discussion

Left circumflex versus right coronary artery stenosis as a cause of posterior infarction. This study clearly demonstrates that patients with electrocardiographic posterior infarction, either alone or more commonly combined with inferior infarction, exhibit more prevalent and more severe circumflex coronary artery disease and more prevalent basal lateral left ventricular asynergy than do patients with only inferior infarction. These findings cast doubt on the common clinical presumption that right coronary artery disease is usually responsible for electrocardiographic posterior infarction (14,15). Probably the main reasons for this presumption are the common occurrence of posterior infarction in conjunction with inferior infarction—as in 17 of our 21 patients—and the belief that the $RV_{1,2}$ electrocardiographic pattern results from necrosis of the basal inferior wall of the left ventricle (5), an area supplied by the right coronary artery. The presumed causal association between electrocardiographic posterior infarction and right coronary disease, however, cannot be reconciled with the markedly lower prevalence of posterior (1 to 24%) as compared with inferior (40 to 60%) or anterior (21 to 74%) infarction in large unselected series (4,9,14–17). In addition, at least two pathologic studies (3,4) have shown that necrosis of the basal inferior wall is common but is rarely associated with electrocardiographic evidence of posterior infarction.

More recently, three separate studies, using different methods, have provided strong evidence that circumflex coronary artery disease is associated with electrocardiographic posterior infarction as manifested by the $RV_{1,2}$ pattern. In a series of 300 patients (6), 65 with normal left ventricular wall motion by radionuclide ventriculography and 235 with single or multiple areas of left ventricular asynergy, nonparametric mathematic analysis showed that the $RV_{1,2}$ electrocardiographic pattern of posterior infarction was most strongly associated with asynergy of the basal lateral left ventricular wall, an area supplied predominantly by the circumflex artery. In a series of 20 patients with isolated posterolateral infarction at autopsy (8), the most common electrocardiographic finding was the $RV_{1,2}$ pattern, seen in 9 patients. Finally, from yet another perspective, in a series of 84 patients with isolated circumflex coronary artery disease (7), 43 patients exhibited the $RV_{1,2}$ pattern of posterior infarction, whereas only 32 had inferior pathologic Q waves and only 2 had lateral Q waves.

Coronary anatomy and disease in posterior infarction. Any hypothesis regarding coronary artery anatomy and disease in electrocardiographic posterior infarction must be compatible with the following facts: 1) Electrocardiographic posterior infarction most commonly occurs in combination with inferior infarction (inferoposterior infarction). 2) Inferoposterior or isolated posterior infarction is far less prevalent than isolated inferior infarction. 3) Circumflex coronary artery disease and left ventricular lateral wall asynergy are strongly associated with the $RV_{1,2}$ pattern of electrocardiographic posterior infarction. Several known patterns of coronary artery anatomy appear to meet these requirements, and their prevalence in our patients with electrocardiographic posterior or inferoposterior infarction can be determined directly from Table 1.

Patients with a dominant or codominant circumflex artery fulfill these requirements because this pattern of coronary anatomy occurs in only about 10% of the population and because a single stenosis or occlusion in such a vessel could readily cause simultaneous infarction of the lateral and inferior walls. This pattern was observed in only 3 of our 21 patients with posterior infarction (Cases 2, 4 and 6, Table 1), all of whom had concomitant inferior infarction, and in 2 of the 21 patients with isolated inferior infarction (Cases 30 and 40). A second and less common variant of coronary anatomy that fulfills these requirements is that of a superdominant right coronary artery giving off lateral left ventricular branches in the area usually supplied by distal marginal branches of the circumflex artery. This pattern was observed in only one patient with inferoposterior infarction (Case 5) and one patient with isolated inferior infarction (Case 4).

Mechanisms of posterior infarction. In most of the patients with posterior or inferoposterior infarction (17 of 21), these less common variations of coronary anatomy were not present, and the only remarkable distinguishing feature of this group was the unusually high prevalence and severity of circumflex artery disease when compared with findings in patients with isolated inferior infarction. These data strongly suggest that inferoposterior infarction most commonly occurs in that subset of patients with right coronary artery disease who also have unusually severe circumflex artery disease. Why this occurs is not known, but it is possible that the following physiologic mechanisms are operative. If the diseased right coronary and circumflex arteries were extensively interconnected by collateral vessels, occlusion of either artery might cause infarction not only in its own distribution but also partially in the distribution of the other artery. Our angiographic data on collateral circulation cannot be used to evaluate this hypothetical mechanism because it cannot be assumed that the postinfarction and preinfarction collateral channels were identical. However, in patients without intercoronary collateral vessels, decreased coronary artery perfusion and increased myocardial oxygen consumption due to an extensive infarction in either the right or circumflex coronary artery distribution could potentially produce sufficiently severe hypotension or left ventricular dysfunction to cause simultaneous infarction in the distribution of the other severely diseased but nonoccluded artery.

Conclusions. Speculation aside, our data clearly show that patients with posterior or inferoposterior infarction have more prevalent and more severe circumflex artery disease than do patients with inferior infarction alone. Because there is currently much emphasis on the use of noninvasive methods to identify postinfarction patients with more extensive coronary artery disease and greater risk of mortality (18), it is useful for the clinician to realize that the simple observation of the $RV_{1,2}$ pattern in patients with inferior infarction is highly predictive of at least two vessel coronary artery disease.

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